

RESULTS Overall, the frequency of CAAs was 272/10,457 (2.60%), including 64 (0.62%) anomalous origin of coronary artery, 180 (1.72%) anomalies of intrinsic coronary arterial anatomy, 24 (0.23%) coronary artery fistula, and 4 (0.04%) number anomalies of coronary artery. Among anomalous origin of coronary artery, RCA originating from the left sinus of Valsalva (n=27, 0.26%), high take-off of the RCA (n=14, 0.13%) and LCX originating from the right sV (n=12, 0.11%) were the top three types of anomalies. Among anomalies of intrinsic coronary arterial anatomy, the frequencies of hypoplasia is 1.24%, aneurysms 0.41%, and coronary atresia 0.01%. No gender differences were presented in the frequencies of most CAAs, except that LCX originating from the right sinus of Valsalva occurred more frequently in males than females (0.11% vs. 0.01%, $p=0.027$). Furthermore, clinical relevance based classifications of CAAs were similar in the males and females.

CONCLUSIONS 320-slice computed tomography can serve as a non-invasive technique for clinical detection of CAAs.

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Mesenchymal Stem Cells Engineered with Integrin-Linked Kinase Improves Cardiac Function Paralleled with Enhanced Homing Capacity Following Intracoronary Transplantation in Porcine Myocardial Infarction

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OBJECTIVES Mesenchymal stem cell (MSCs)-based therapy is under investigation for treating acute myocardial infarction (MI) but is limited due to poor engraftment and limited regenerative potential. Integrin-linked kinase (ILK) overexpression enhances progenitor cells homing, promotes cardiomyogenesis, reverses myocardial remodeling and improves cardiac function. We sought to engineer MSCs with ILK and evaluate their therapeutic potential on acute MI in a large-animal model supposing a synergistic effect of MSC and ILK could be achieved.

METHODS Minipigs underwent a 90-minute balloon occlusion of the left anterior descending coronary artery followed by reperfusion. ILK-engineered MSCs were intracoronary transplanted 7±1 days after occlusion, as were vector-engineered MSCs and vehicle controls. Both cells were GFP- and iron-labeled before transplantation and were monitored *in vivo* by cardiac magnetic resonance imaging (CMR). CMR was also used to measure global and regional left ventricular (LV) function, scar size and perfusion.

RESULTS No impairment occurred in biological properties of MSCs following iron labeling at 50µg/ml. Significantly enhanced homing capacity of MSCs following ILK modification was revealed by MRI *in vivo* and confirmed by histological staining. Intracoronary transplantation of ILK-engineered MSCs improves global LVEF by 7.8% compared with baseline ($P=0.03$), and 11.7% when compared with vehicle ($P=0.02$) 15 days post-implantation. Regional LV contractile function was also recovered ($P<0.001$), accompanied by substantially reduced scar size ($P<0.001$), myocardial remodeling ($P<0.05$), cell apoptosis ($P<0.001$), and increased regional perfusion ($P<0.001$) and cardiac cell proliferation ($P<0.001$) in ILK-MSC treated minipigs versus vehicle controls. Vector-engineered MSC did not produce significant improvement in global LVEF or reduction in cardiac remodeling compared with baseline or vehicle controls, and generated less extent of all other favorable effects compared with ILK-modified MSCs.

CONCLUSIONS ILK-engineered allogeneic MSCs enhance regional and global LV function, reverse the remodeling process and restore regional perfusion with an improved homing capacity following intracoronary transplantation. Engineering MSCs with ILK has great potential implication for cell therapy in post-MI patients.

GW26-e1077

Impact of body mass index on mortality in patients with diabetes: Meta-analysis of 20 studies including 250,016 patients

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OBJECTIVES The influence of body weight on mortality among patients with diabetes remains controversial. Therefore, we performed a meta-analysis of pertinent studies.

METHODS We searched OVID/MEDLINE, EMBASE and Cochrane databases for all reported studies, published before Dec 2014, which investigated the relationship between body mass index (BMI) and mortality in patients with diabetes. Summary estimates of hazard ratios (HRs) were obtained with a random effects model. Univariate meta-regressions were performed.

RESULTS Twenty studies including 250,016 patients with diabetes were identified. The meta-analysis demonstrated a significantly reduced risk of all-cause mortality in overweight patients (HR 0.82, 95% CI 0.74 to 0.91, $P<0.0001$, and $I^2=91.6\%$) as compared to normal weight patients. The survival benefits of obesity was only observed in the elderly patients (HR 0.69, 95% CI 0.63 to 0.75, $P<0.0001$, and $I^2=50.4\%$), but not in the younger patients (HR 1.01, 95% CI 0.84 to 1.20, $P=0.96$, $I^2=80.1\%$). Moreover, meta-regression analysis indicated that the beneficial prognostic impacts on overweight (Coefficient=0.030; $p=0.041$) and obesity (Coefficient=0.032; $p=0.010$) were attenuated with clinical follow-up duration.

CONCLUSIONS Our meta-analysis indicated a significantly lower risk of mortality in overweight patients with diabetes compared to normal weight patients. However, the survival benefits of obesity were only observed among the elderly patients.

GW26-e2187

Association between Genetic Variation in NFKB1 and NFKBIA and Susceptibility to Coronary Artery Disease in a Chinese Han Population

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OBJECTIVES Coronary artery disease (CAD) is a chronic inflammatory disease involving the complex interplay between multiple genetic and environmental factors. As a central regulator of inflammation, NF-κB plays an important role in the development and progression of inflammatory diseases. The aim of this study was to investigate whether promoter polymorphisms in NFKB1 and NFKBIA gene contribute to CAD in a Chinese Han population.

METHODS This is a case-control study. NFKB1 promoter polymorphism (-94ins/del ATTG) and NFKBIA promoter polymorphisms (-881A/G, -826C/T, and -297C/T) were genotyped using TaqMan SNP genotyping assays in 1140 Han CAD cases and 1156 Han CAD-negative controls, and then NFKBIA haplotype blocks were reconstructed according to our genotyping data.

RESULTS No statistical significance was observed for the distribution frequency of the NFKBIA -881 A/G, -826 C/T, or -219 C/T allele, genotype and haplotype polymorphisms between CAD cases and controls. None of the studied NFKBIA SNPs were associated with CAD. There was significant difference in the distribution of the genotypes ($P=0.001$) and alleles ($P=0.001$) of NFKB1-94ins/del polymorphism in CAD cases and controls. The homozygous variant genotype of NFKB1-94ins/del ATTG was consistently associated with increased risk of CAD among all participants after adjustment for covariates (OR=1.505, 95% CI 1.190 to 1.903, $P=0.001$).

CONCLUSIONS In our study, we did not detect any relationship between NFKBIA promoter polymorphisms and CAD. However, the -94 ins/del polymorphism in NFKB1 promoter affects the susceptibility for CAD in Chinese Han population, providing a new insight into the genetic basis of CAD in Chinese Han population.

GW26-e1324

Reduced T-cell thymic export reflected by sj-TREC in patients with coronary artery disease

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OBJECTIVES Thymus involution can contribute to immune disturbance. This study aimed to explore whether recent thymic export